A CASE OF KERATODERMIA PUNCTATA

BY

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Keratodermia punctata is a very rare disease. The following case is a good example of it and fulfils the criteria for diagnosis postulated by Sweitzer (1923), who stated that "the true keratoderma punctatum should not show a generalized hyperkeratosis or any erythema at the edges of the lesions." Cleveland White (1930) had a similar case treated by chiropody for twelve years without improvement. My patient had been treated by a chiropodist for two months without success.

Case Report

A gunner aged 32 was admitted to a general hospital in B.L.A with a provisional diagnosis of "? multiple verrucae of hands." After examination and investigation keratodermia punctata was diagnosed. The patient had had no previous illnesses or skin disease, and had always been healthy. He was a farmer in civil life and had had four years' service as an infantryman before joining the artillery. The family history was completely negative.

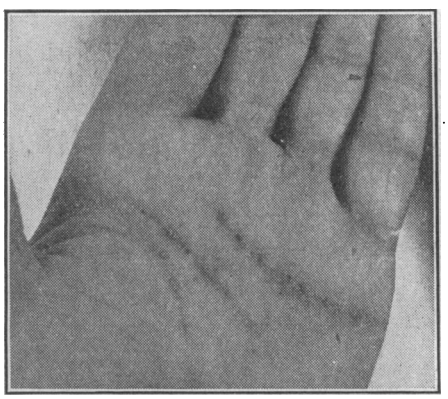

FIG. 1.—Photograph showing crateriform pits, some of which are filled with horny plugs.

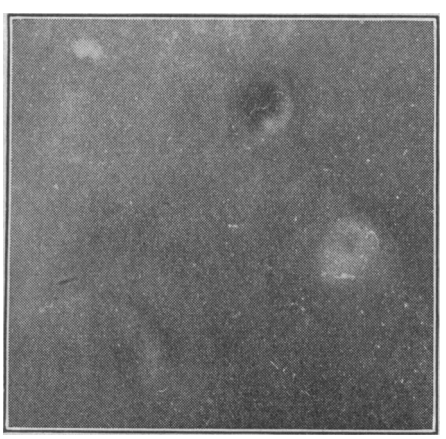

FIG. 2.—Enlarged photograph of actual lesions.

Three months before admission he noticed small hard "lumps" developing in the creases of his palms and on the inner borders of his feet. There was slight pain on walking and on pressure, and the lesions were described by the patient as "plugs which remained for about a week, then dropped out, and new plugs developed." There was no history of his having taken arsenic or any other drug. He was of good physique, and nothing abnormal was discovered in any of the systems. The urine was normal; the Kahn test negative. The general skin condition was normal.

Hands.—Multiple small crateriform pits, some empty and some filled with greyish-black horny plugs, were found, entirely limited to the creases of the palms. Generalized hyperkeratosis was not present. Each lesion was surrounded by normal skin and there was no erythema. On the left hand the majority of the lesions were situated in the two distal creases. There were fewer lesions on the right hand, and a number of grouped puncta were situated at the inner end of the distal crease. This group was removed for a biopsy and the report stated: "Hyperkeratosis only. No inflammatory changes present."

Feet.—A few grouped lesions were found on the inner borders of the soles of the feet. The skin apart from these lesions was normal. There was no hyperkeratosis, clavus, or other abnormality.

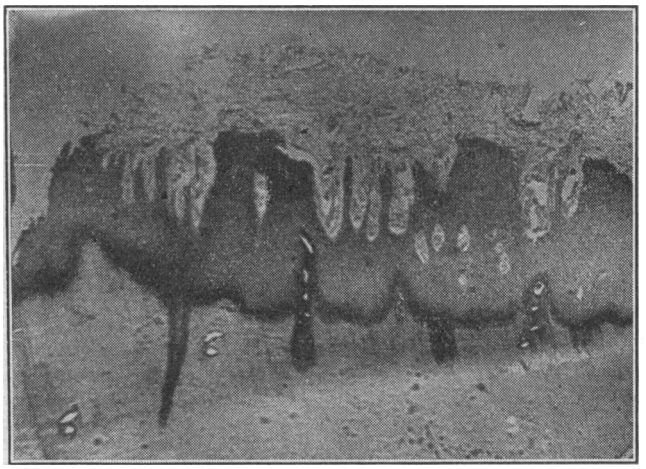

FIG. 3.—Photomicrograph of one of the lesions, showing hyperkeratosis only.

The patient was treated with a keratolytic application—ung. acid. salicyl., 15%, applied thrice daily—with some improvement. He was returned to duty as there was little or no disability.

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VITAMIN K FOR THE RELIEF OF CHILBLAINS

BY

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Chilblains consist of a reaction to cold in susceptible persons. The predisposing factors in such people are assumed to be defective peripheral circulation, with increased permeability of the vessel walls and diminished coagulability of the blood. Such is the description given by MacKenna (1937). It would seem logical, therefore, to treat such a condition with a substance which will correct these abnormalities. In vitamin K we have such a substance, for in avitaminosis K the same abnormalities are present and are corrected by the administration of the vitamin.

Stewart and Rourke (1939) showed that hypoprothrombinaemia results from lack of vitamin K and that the prothrombin level in such cases could be restored to normal by the vitamin. Scarborough and Macfarlane (1942) pointed out that hypoprothrombinaemia gave rise to a delayed coagulation time, and concluded that a capillary defect might also be present in that condition. A somewhat similar state of affairs occurs in the allergic manifestation known as urticaria. Black (1945) studied the prothrombin level in a number of cases of chronic urticaria and found it diminished in 65%. He also found that the condition could be relieved by vitamin K therapy, the most marked improvement, in fact, occurring in those cases with lowered prothrombin level.

On these considerations is based the following small series of cases. Unfortunately, it was not possible to estimate

the prothrombin times in these cases; and the results are based on clinical observations alone. The prolonged spell of severe weather last winter afforded a unique opportunity for assessing the efficacy of the treatment. A commercial preparation of acetomenaphthone, synthetic vitamin K, was used throughout.

Case Reports

Case 1.—A man aged 37 had suffered from chilblains ever since he could remember. He was engaged on outdoor work, which aggravated them. He had tried numerous remedies, including calcium in various forms, a variety of ointments, and a full course of concentrated vitamin D injections, without effect. All his fingers and toes were swollen and dusky red, with signs of ulceration on several of the toes. He was given a single intramuscular injection of 5 mg. of acetomenaphthone. When seen one week later he was ecstatic, saying that for the first time he had obtained relief from his symptoms. His fingers were normal, and the toes showed only a slight residual swelling. A course of 10 mg. orally twice daily was instituted. By the end of a further week all trace of chilblains had disappeared. He was not seen again for two months, when he came up voluntarily to express his admiration of the treatment. During this time he had received no further vitamin K and had continued with his outdoor work. The chilblains had not recurred, and on examination there was no sign of any. The only symptom was a slight tingling in the toes of a few days' duration; this rapidly disappeared with a further course of the drug.

Case 2.—A Continental married woman refugee aged 30 had never had chilblains before coming to England. Now she has them all the year through, in a state of continual ulceration. She had tried all the usual treatments—calcium, vitamin D by mouth and injection, and injection of concentrated vitamin D, etc.—with no effect. All her fingers were grossly swollen, reddened, and ulcerated at most of the joints. She was given 5 mg. of synthetic vitamin K by intramuscular injection. Eight hours later the swellings had greatly subsided, although the ulcerations remained unaffected. Unfortunately the injection caused a great deal of pain, and she declined to have more. Instead, a course of 20 mg. orally twice daily was instituted. The patient was seen two weeks later, when there was some duskiness of the fingers and slight ulceration. The swellings, however, had completely subsided and the remaining ulceration was far less than it had been. In addition, there was now no irritation. A month later, however, during which time she had continued treatment, the fingers had again ulcerated and were again irritable; this coincided with the severe weather conditions. Nevertheless, the lesions were not nearly so bad as they had been at first, when the weather had not been particularly cold.

Case 3.—A married woman aged 28 had for the last seven years suffered from chilblains on her toes each winter. Last winter they had come on with particular severity and had invaded her heels and even the backs of her legs. They were present most of that season, with acute exacerbations during cold spells. When seen she was undergoing one of these exacerbations, and the back of the lower half of each leg was covered with several large, raised, reddened chilblains. They were not ulcerated, but were giving rise to intense irritation. She was given 20 mg. of acetomenaphthone thrice daily; the first dose was taken in the morning, and by the evening the irritation had ceased. Previously this had been constantly present. She was seen four days later, during which time there had been no irritation, and she was highly delighted with the result. The chilblains on her legs were still present, although there was a definite diminution in redness and swelling. Treatment was continued, and for a week she was free of symptoms. Then, however, the severe cold set in and the irritation started again, although there was no swelling, only dusky bluish patches in the skin. She was advised to continue with the treatment in spite of this setback. Unfortunately, she failed to attend again, and so further results were not known.

Case 4.—Personal observations. In this case mild chilblains occur in cold weather only. They never ulcerate, but give rise to intense irritation, most pronounced on going from a cold atmosphere to a warm one. They always clear up with milder weather. The patient, being somewhat averse to intramuscular injections, had an oral course of 20 mg. of vitamin K daily. After five days of this the chilblains had disappeared. Unfortunately this test coincided with a change in the weather, so it was not conclusive. Soon afterwards, however, the weather again became very cold, and this spell lasted for several months, and so it was possible to make more conclusive observations. To begin with, 20 mg. were taken twice daily before the chilblains had had time to develop. This did not prevent their appearance, but their onset occurred much more slowly and with much less irritation than before. After five days the doses were increased to thrice daily, and after a further five days all irritation had ceased and the swellings had almost subsided. The doses were decreased to twice daily as before, and no further trouble was experienced for three weeks. At the end of this time the irritation restarted, so doses were again taken thrice daily and the symptoms cleared in a few days. A maintenance dose of 10 mg. daily proved insufficient to prevent the recurrence of symptoms within a few days. Again these were cured by the larger doses, which were continued for the rest of the cold weather—about five weeks, during which time no further symptoms were encountered.

Case 5.—A married woman aged 21 had some moderately severe chilblains on her toes and heels last winter for the first time. These cleared up after a few days' treatment with vitamin K (20 mg. twice daily) and did not recur on cessation of treatment.

Case 6.—A woman aged 21 had had chilblains on her toes and heels every winter for the past four years. They were of moderate severity, but gave rise to such intense irritation that she dared not go very close to a fire for fear of aggravating them. She was sceptical that any treatment could give relief, but, to her surprise, after one week of vitamin K (20 mg. twice daily) her symptoms were greatly relieved and the swellings much reduced. Treatment was continued, but the patient failed to keep any further appointments.

Case 7.—A French girl aged 19 had suffered from chilblains since coming to this country several months previously. She had never had them before, and ascribed them to her inadequate diet during the German occupation. Her fingers were affected and were much swollen, but her toes were free. After one week's treatment (20 mg. twice daily) there was little improvement. For the second week bile salts were administered with the vitamin K, and there was now some improvement, the swellings being smaller and bluer. There was, however, no further improvement at the end of a third week, although she considered her symptoms to be somewhat better.

Case 8.—A man aged 46 occasionally suffered from chilblains in the winter. When seen he had moderate chilblains, together with infected corns, on his toes only. He was given 20 mg. of vitamin K twice daily and his chilblains disappeared in three to four days and did not recur, although he took only one week's course of the treatment.

Discussion

In this small series of cases the administration of vitamin K would definitely seem to have exerted a favourable influence on the condition known as chilblains. The dosage appears to vary from individual to individual, and is not related to the severity of the condition. On an average a dose of 20 mg. twice daily was found most generally useful. Again, in some cases a short course of treatment was sufficient to give protection for a long period, whereas in others it was necessary to continue the dosage to prevent recurrence of the lesions.

Howell (1941) suggested that prothrombin and fibrinogen might be constantly formed and consumed in the blood, the result being the formation of fibrin, which in some way might provide nourishment or protection for the cells of the capillary walls. It was on this assumption that the work on urticaria was done.

Might it not be that in chilblains there is a similar impairment of this mechanism, with resultant damage to the capillary walls—the cause in this case being cold rather than allergic stimulus? The individual susceptibility might

be explained by a mild hypovitaminosis-K in certain individuals. Kark and Lozner (1939) showed that this hypovitaminosis could in fact occur in adults owing to causes other than liver disease. In any case it would seem worth while giving vitamin K a further trial in this widespread and irritating condition.

Summary

The treatment of eight cases of chilblains, ranging from the mildest manifestation to severe and ulcerated forms, is described. In four there was complete alleviation of symptoms and signs, the response in Case 1 being exceptionally good. In the other four there was an improvement. Two of these patients defaulted before treatment was completed, so the final result was not known. In the remaining two there was improvement, especially of the actual chilblains, although the perniotic condition of the digits remained; and in one there was a recrudescence of symptoms in spite of continued treatment.

As regards the mode of administration it was considered that intramuscular injections were superior to the oral route. They were, however, accompanied by considerable pain, so they had to be abandoned, as the patients were not prepared to put up with this for the possible cure of their relatively minor ailment.

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CALCIUM DEPOSITS IN THE IRIS IN A CASE OF SECONDARY HYPERPARATHYROIDISM

BY

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The following case is of interest for two reasons: (*a*) a review of the literature does not reveal any previous reference to such deposits in these cases; (*b*) renal calculi and metastatic calcification were absent from the kidneys.

Oliver (1939) refers to three cases of hyperparathyroidism, as follows. (i) Dawson and Struthers (1923). A man aged 49 with a long history of multiple fractures, ending in syncope and death; necropsy showed a parathyroid tumour, calcification of vessels (heart, lungs, kidney, liver, spleen). (ii) Lowenburg and Ginsberg (1932) describe the case of a boy aged 5 who received an overdose of parathormone, 100 units daily for 6 days, with resultant vomiting and listlessness after two days, followed by mental depression and high fever. Blood examination on the sixth day showed serum calcium 19.6 and blood phosphorus 4.4 mg. per 100 ml. On withdrawal of the drug the patient became normal. (iii) Hanes (1939) mentions a woman aged 49 who complained of weakness, pains in the legs, and loss of weight. Previous radiography of the kidneys, when in hospital for pyelitis, had shown diffuse fine mottling. Serum calcium was 12 mg. per 100 ml. and renal functions were markedly impaired. Five years later, on final admission to hospital, she had a tumour of the lower pole of the thyroid. Blood calcium was 20 mg., phosphorus 4.7 mg., phosphates 23 mg., and non-protein nitrogen 58 mg. per 100 ml. Skiagrams showed metastatic calcification in the kidneys and decalcification of bones. The patient became weak, depressed, and febrile, and she soon died. Necropsy showed a necrotic partly calcified parathyroid tumour.

Moore (1943) states that calcification had been reported in many organs in hyperparathyroidism, but not in bone-marrow. He mentions calcification in kidney, stomach, heart, liver, and marrow found in experiments on animals. Werner (1942) describes metastatic deposits in the kidney, and says they can occur in other organs and tissues (none mentioned specifically). He also quotes Allbright, Baird, Cope, and Bloomberg for renal calculi; and Dawson and Struthers as finding calcium infiltration in practically every organ of the body. Jaffe (1940) states that secondary parathyroid hyperplasia is found more regularly in connexion with chronic renal insufficiency than in any other condition, and that the hyperfunction becomes severe enough to induce pronounced skeletal and other tissue changes, the chief lesions being skeletal and renal (calculi). Nephrosclerosis is less common. Severe renal insufficiency is followed by calcification in soft parts (subcutaneous tissues and arteries).

Case History

The patient had a history of having been taken ill in October, 1944, while serving in the East. At that time he complained of loss of weight, loss of appetite, frequency of micturition, and looseness of the teeth. He was admitted to hospital, where the urine was found to be of low specific gravity and to contain albumin and hyaline and granular casts. Skiagrams of the kidneys, after intravenous "uroselectan," failed to reveal the outline of any renal tissue. The blood urea was 140 mg. per 100 ml.

In November, 1944, he became drowsy and the skin dry. At this period he developed a left-sided Bell's palsy; and the ear-drums were found to contain chalky deposits. Soon afterwards vision became blurred, and examination revealed a chalky deposit in the anterior chamber of the right eye and retina—choroidal atrophy. By this time the rough skin showed nodularity, which appeared to be due to chalk-like deposits. Blood examination revealed a serum calcium of 17 mg. and inorganic phosphorus of 4.3 mg. per 100 ml.; the phosphate index was normal. Radiology of the long bones showed widespread osteoporosis.

By February, 1945, the serum phosphate was 22 units (normal 3–13), and a diagnosis of hyperparathyroidism was made. Blood calcium and blood urea could be controlled temporarily by reducing intake of phosphates. The blood calcium fell to 11 mg. and the blood urea to 82 mg. per 100 ml., only to rise again in two weeks. Primary hyperparathyroidism was considered to be the diagnosis.

In June, 1945, the urine volume was 3½ pints (2 litres) daily, S.G. 1012 or less. Urea clearance was 30% of normal, and the blood urea 80 mg. per 100 ml. *X*-ray investigation showed well-marked calcification of the peripheral vascular tree in places. His general condition had improved, although wasting of the legs was extreme, and the diagnosis of primary hyperparathyroidism was thought to be incorrect because of the normal serum phosphates, absence of urinary calculi, and absence of skeletal weakness (neither fractures nor bowing of long bones).

In February, 1946, the patient was admitted to Childwall Hospital for review. On admission he complained of loss of balance when walking. He was emaciated and there were calcium deposits beneath the nails. Disks and ear-drums were as on previous occasions. In addition the irises showed fine deposits of calcium. Urea concentration was 1.3%, and faeces calcium 60% (normal reading 80% to 90%). Skiagrams of arteries showed slight calcification in the walls of the arteries of both hands, and those of the skull, spine, and long bones revealed generalized slight osteoporosis. Blood calcium amounted to 14 mg., blood urea 52 mg., serum albumin 4.5 mg., serum globulin 1.5 mg., and inorganic phosphate 3.5 mg. per 100 ml. The phosphate index was normal; there was a secondary anaemia with moderate eosinophilia. Examination of the urine showed protein 1%; sodium chloride, 6.5 mg. in 24 hours; calcium oxalate ++, hyaline casts +, and a few granular casts; red cells were occasionally present.

Progress of the Case.—Owing to the emaciation present it was considered advisable to put the patient on a fuller diet about